# abcam

### Product datasheet

## PE Anti-Tau antibody [EP2456Y] ab210985

Recombinant RabMAb

#### 2 Images

#### Overview Product name PE Anti-Tau antibody [EP2456Y] Description PE Rabbit monoclonal [EP2456Y] to Tau Host species Rabbit Conjugation PE. Ex: 488nm, Em: 575nm Specificity ab76128 detects Tau, whether phosphorylated or unphosphorylated on serine 635. The specificity of this antibody refers to P10636-8. This antibody clone has been shown to react with Tau 4R and 3R isoforms (doi.org/10.1101/2023.04.13.536711). Our testing suggests that this antibody clone does not cross-reacts with MAP2 or MAP4 **Tested applications** Suitable for: Flow Cyt (Intra) **Species reactivity** Reacts with: Mouse Predicted to work with: Rat, Human Synthetic peptide within Human Tau aa 600-700 (C terminal) (phospho S305). The exact Immunogen immunogen sequence used to generate this antibody is proprietary information. If additional detail on the immunogen is needed to determine the suitability of the antibody for your needs, please contact our Scientific Support team to discuss your requirements. Database link: P10636-8 **Positive control** Flow Cyt (intra): Neuro 2A (differentiated) cells **General notes** Our RabMAb<sup>®</sup> technology is a patented hybridoma-based technology for making rabbit monoclonal antibodies. For details on our patents, please refer to RabMAb® patents.

Properties	
Form	Liquid
Storage instructions	Shipped at 4°C. Upon delivery aliquot. Store at +4°C. Do Not Freeze. Store In the Dark.
Storage buffer	pH: 7.4
	Preservative: 0.02% Sodium azide
	Constituents: PBS, 1% BSA

Purity	Protein A purified
Clonality	Monoclonal
Clone number	EP2456Y
Isotype	lgG

#### Applications

The Abpromise guarantee Our <u>Abpromise guarantee</u> covers the use of ab210985 in the following tested applications.

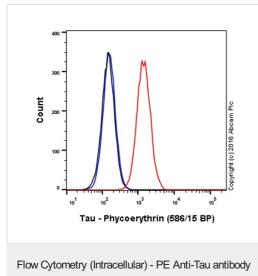
The application notes include recommended starting dilutions; optimal dilutions/concentrations should be determined by the end user.

Application	Abreviews	Notes
Flow Cyt (Intra)		1/5000. The cellular localisation of this product has been verified in ICC/IF

Function	Promotes microtubule assembly and stability, and might be involved in the establishment and maintenance of neuronal polarity. The C-terminus binds axonal microtubules while the N-terminus binds neural plasma membrane components, suggesting that tau functions as a linker protein between both. Axonal polarity is predetermined by tau localization (in the neuronal cell) in the domain of the cell body defined by the centrosome. The short isoforms allow plasticity of the cytoskeleton whereas the longer isoforms may preferentially play a role in its stabilization.
Tissue specificity	Expressed in neurons. Isoform PNS-tau is expressed in the peripheral nervous system while the others are expressed in the central nervous system.
Involvement in disease	<ul> <li>Note=In Alzheimer disease, the neuronal cytoskeleton in the brain is progressively disrupted and replaced by tangles of paired helical filaments (PHF) and straight filaments, mainly composed of hyperphosphorylated forms of TAU (PHF-TAU or AD P-TAU).</li> <li>Defects in MAPT are a cause of frontotemporal dementia (FTD) [MIM:600274]; also called frontotemporal dementia (FTD), pallido-ponto-nigral degeneration (PPND) or historically termed Pick complex. This form of frontotemporal dementia is characterized by presenile dementia with behavioral changes, deterioration of cognitive capacities and loss of memory. In some cases, parkinsonian symptoms are prominent. Neuropathological changes include frontotemporal atrophy often associated with atrophy of the basal ganglia, substantia nigra, amygdala. In most cases, protein tau deposits are found in glial cells and/or neurons.</li> <li>Defects in MAPT are a cause of Pick disease of the brain (PIDB) [MIM:172700]. It is a rare form of dementia pathologically defined by severe atrophy, neuronal loss and gliosis. It is characterized by the occurrence of tau-positive inclusions, swollen neurons (Pick cells) and argentophilic neuronal inclusions known as Pick bodies that disproportionally affect the frontal and temporal cortical regions. Clinical features include aphasia, apraxia, confusion, anomia, memory loss and personality deterioration.</li> <li>Note=Defects in MAPT are a cause of corticobasal degeneration (CBD). It is marked by extrapyramidal signs and apraxia and can be associated with memory loss. Neuropathologic features may overlap Alzheimer disease, progressive supranuclear palsy type 1 (PSNP1) [MIM:601104, 260540]; also abbreviated as PSP and also known as Steele-Richardson-Olszewski syndrome.</li> </ul>

Form	There are 9 isoforms produced by alternative splicing.
Cellular localization	Cytoplasm > cytosol. Cell membrane. Cytoplasm > cytoskeleton. Cell projection > axon. Mostly found in the axons of neurons, in the cytosol and in association with plasma membrane components.
	<ul> <li>Phosphorylation decreases with age. Phosphorylation within tau's repeat domain or in flanking regions seems to reduce tau's interaction with, respectively, microtubules or plasma membrane components. Phosphorylation on Ser-610, Ser-622, Ser-641 and Ser-673 in several isoforms during mitosis.</li> <li>Polyubiquitinated. Requires functional TRAF6 and may provoke SQSTM1-dependent degradation by the proteasome (By similarity). PHF-tau can be modified by three different forms of polyubiquitination. 'Lys-48'-linked polyubiquitination is the major form, 'Lys-6'-linked and 'Lys-11'-linked polyubiquitination also occur.</li> <li>Glycation of PHF-tau, but not normal brain tau. Glycation is a non-enzymatic post-translational modification that involves a covalent linkage between a sugar and an amino group of a protein molecule forming ketoamine. Subsequent oxidation, fragmentation and/or cross-linking of ketoamine leads to the production of advanced glycation endproducts (AGES). Glycation may play a role in stabilizing PHF aggregation leading to tangle formation in AD.</li> </ul>
modifications	kinases (PDPK: CDK1, CDK5, GSK-3, MAPK) (only 2-3 sites per protein in interphase, seven- fold increase in mitosis, and in PHF-tau), and at serine residues in K-X-G-S motifs by MAP/microtubule affinity-regulating kinase (MARK) in Alzheimer diseased brains.
Domain Post-translational	The tau/MAP repeat binds to tubulin. Type I isoforms contain 3 repeats while type II isoforms contain 4 repeats. Phosphorylation at serine and threonine residues in S-P or T-P motifs by proline-directed protein
Developmental stage	Four-repeat (type II) tau is expressed in an adult-specific manner and is not found in fetal brain, whereas three-repeat (type I) tau is found in both adult and fetal brain.
Sequence similarities	Contains 4 Tau/MAP repeats.
	PSNP1 is characterized by akinetic-rigid syndrome, supranuclear gaze palsy, pyramidal tract dysfunction, pseudobulbar signs and cognitive capacities deterioration. Neurofibrillary tangles and gliosis but no amyloid plaques are found in diseased brains. Most cases appear to be sporadic, with a significant association with a common haplotype including the MAPT gene and the flanking regions. Familial cases show an autosomal dominant pattern of transmission with incomplete penetrance; genetic analysis of a few cases showed the occurrence of tau mutations, including a deletion of Asn-613.

#### Images

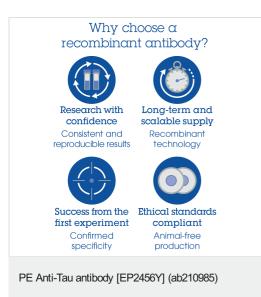


[EP2456Y] (ab210985)

Overlay histogram showing Neuro 2A (differentiated) cells stained with ab210985 (red line). The cells were fixed with 4% formaldehyde (10 min) and then permeabilized with 90% methanol (-20°C) for 30 min. The cells were then incubated in 1x PBS / 10% normal goat serum to block non-specific protein-protein interactions followed by the antibody (ab210985, 1/5000 dilution) for 30 min at 22°C.

Isotype control antibody (black line) was rabbit IgG (monoclonal) Phycoerythrin (<u>ab209478</u>) used at the same concentration and conditions as the primary antibody. Unlabelled sample (blue line) was also used as a control.

Acquisition of >5,000 events were collected using a 50mW Yellow-Green laser (561nm) and 586/15 bandpass filter.



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